Unilateral Paralysis of Eye Muscles Associated with Intracranial Saccular Aneurysms

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SUMMARY

Unexplained unilateral paralysis of eye muscles with or without evidence of altered function of other cranial nerves, or of the presence of intracranial bruit, is strong positive diagnostic evidence of saccular aneurysm of the intracranial internal carotid or of the circle of Willis. If pulsating exophthalmos is present the lesion probably is an arteriovenous fistula.

The presence of such clinical signs strongly indicates the desirability of cerebral angiography. Once demonstrated, the lesion can usually be treated successfully by ligation of the contributing vascular connections.

SUDDEN one-sided paralysis or weakness of any movement of the eyeball should suggest the possible presence of an intracranial saccular aneurysm of the internal carotid artery or one of its major branches. The usual complaint will be of a drooping eyelid or of double vision. Additional symptoms may indicate involvement of other cranial nerves as well. There may be loss of vision in one eye or in both, suggesting pressure on the optic tract or the chiasm. Pain or hypesthesia in the distribution of the trigeminal nerve may be present if this region has been affected; the corneal reflex may be diminished or absent. Should the aneurysm have ruptured, the usual signs of subarachnoid hemorrhage may be found. Occasionally a bruit can be heard, but its absence does not point away from aneurysm as a causative lesion in this syndrome of the region of the cavernous sinus. In our experience the presence of a bruit is unusual.

If there is an associated unilateral exophthalmos, particularly of the pulsating type, an arteriovenous aneurysm between the internal carotid and the cavernous sinus should be suspected. In such circumstances a bruit is usually to be heard.

An untreated aneurysm is liable to rupture, as was demonstrated by McDonald and Korb (1939)[4] who reported such an occurrence in 786 of the 1,125 instances of intracranial aneurysm they found in the literature. The leaking or rupturing of an aneurysm is not necessarily fatal, although the prognosis increases in gravity with each successive bout of intracranial bleeding. Few persons survive the third hemorrhage from such a process. The mortality rate in untreated intracranial aneurysm must be considered to be high.

The possible fate of the patient with undiagnosed and untreated aneurysm of the carotid or of the circle of Willis is illustrated by the history of a woman in her early forties who was said to have been in good health until the onset of the terminal illness, which commenced one evening with the sudden appearance of diplopia. This progressed in a few minutes to complete paralysis of the third nerve, and there was transitory headache on the same side. She was hospitalized for two weeks, during which time the oculomotor palsy showed no improvement, although the headache subsided completely in the first few days. On the night after discharge she was awakened by severe, intolerable generalized head pain. This quieted somewhat, but recurred in about 13 hours, being immediately followed by loss of consciousness, respiratory failure and death.

This patient had a saccular aneurysm of the left carotid artery. The initial symptom was a third cranial nerve paralysis, which even preceded the symptom of subarachnoid hemorrhage. By current methods of study, the outlook in her case should have been better. A presumptive diagnosis of saccular aneurysm would have been made and angiographic studies instituted, followed by definitive therapy directed toward the presumably demonstrable lesion.

In recent years the authors have seen nine patients with saccular aneurysm of the carotid artery or circle of Willis. Angiograms confirmed the diagnosis in each case. In three patients the initial symptom was paralysis of extraocular movement. Involvement of other adjacent cranial nerves, principally the trigeminal, or symptoms resulting from frank subarachnoid bleeding were seen as initial signs in the other six patients.

Two patients were males and seven females. The age at the time of onset ranged from 3 to 53 years. All aneurysms were treated by ligation of the carotid artery in the neck, and in two patients the aneurysm was trapped by clipping the vessel intracranially. The youngest patient, who was 10 at the time of operation, developed hemiparesis 54 hours after carotid ligation. No other undesirable sequelae have been encountered.

Read before the Section on Neuropsychiatry at the 76th Annual Session of the California Medical Association, Los Angeles, April 30-May 3, 1947.

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In all instances oculomotor paralysis was a prominent feature in the clinical diagnosis. To emphasize the importance of this sign, brief histories are given of the three patients in whom it appeared as the initial symptom.

CASE 1. A 53-year-old woman noticed some diplopia, particularly when she was reading, doing a considerable amount of work with her eyes, or when she was tired. This had become progressively worse over a period of eight months. At the same time she noticed a progressive droop of the left eyelid and a tendency for the eye to turn outward.

Approximately six months before entering the hospital she had periodic headaches on the left side of the head, localized behind the left eye. Sometimes the pain was sharp and throbbing; sometimes it was only a dull, retro-orbital and temporal ache. The headaches were not continuous, but occurred almost every day and would last for several hours. The patient was treated for migraine. Frank double vision was recognized by the patient at about the same time that the headaches appeared. This situation continued up to the time of her admission for study. On examination, the striking observations were the ptosis of the left eyelid, the weakness of all movements related to oculomotor nerve function, and a generalized hypalgesia of all divisions of the fifth cranial nerve, but particularly of the ophthalmic branch. A diagnosis of aneurysm of the carotid artery or the circle of Willis on the left side was made and angiographic studies were carried out.

The resulting film showed a saccular aneurysm arising from the cavernous portion of the carotid artery in a position where it could be expected to cause pressure upon the third and fifth cranial nerves. Tying of the internal and external carotids in the cervical region did not relieve the patient's oculomotor palsy which progressed to a complete lesion; therefore a trapping of the aneurysm by intracranial approach was deemed necessary.

At operation there was found to be pressure on the third cranial nerve before it entered the wall of the cavernous sinus and the anterior clinoid. The artery and the optic nerve were displaced mesially. Tantalum clips were placed just proximal to the middle and anterior cerebral arteries. Postoperatively the patient's course was satisfactory. There was no loss of vision in the left eye.

CASE 2. A 43-year-old woman had diplopia of sudden onset without apparent cause two years before examination. She observed that this was worse on gaze to the left. Eleven months later she noted drooping of the right upper eyelid. Two months before admission maxillary pain on the right appeared and progressed in the following weeks to an excruciating degree.

On examination paralysis of all extraocular movement was found. There was anesthesia of the upper portion of the face on the right side and hypesthesia of the lower portion. The muscles of mastication were paralyzed on the right.

Plain roentgen films showed erosion of the base of the skull, and carotid angiography demonstrated a saccular aneurysm just proximal to the cavernous sinus to be the cause of the difficulty. Ligation of the internal carotid was carried out with satisfactory results.

CASE 3. A woman 54 years of age suddenly developed double vision 16 months before hospitalization. This persisted and an unsuccessful attempt was made to correct it with glasses. One month before admission she noted progressively increasing pain in the region behind the right eyeball. Angiography demonstrated abnormal narrowing and displacement of the carotid. Craniotomy was performed and an aneurysm at the site of vascular distortion was trapped between ligation in the neck and clips distal to the defect intracranially.

DISCUSSION

In recent years the problem of saccular aneurysm of intracranial vessels has increasingly commanded the attention of neurologists throughout the world. Although the problem has been the subject of discussion in the literature for over 150 years, aneurysms were still a clinical rarity at the close of the first World War. Only in the past 20 years, since the pioneer work on cerebral angiography by Egaz Moniz, has any consistency in diagnosis and treatment been developed. Today a high percentage of these potentially fatal lesions, particularly those in the anterior part of the circle of Willis and its immediate branches, can be alleviated if recognized clinically, confirmed by angiography and treated early. The importance of unilateral extraocular muscle paralysis as a localizing sign for aneurysms in this region has been stressed by others. Its importance as a diagnostic sign would seem to justify further emphasis.

A discussion of the anatomical basis for the involvement of the oculomotor, trochlear and abducens nerves by aneurysmal lesions in adjacent vessels may serve to emphasize the importance of eye signs.

The internal carotid artery enters the skull by way of the carotid canal in the petrous portion of the temporal bone where it becomes surrounded by a projection of dura mater. Passing beneath the trigeminal nerve in Meckel's cave, it curves cephalad and forward lying within the cavernous sinus and close to the lateral aspect of the body of the sphenoid. As it swings upward toward the mesial aspect of the anterior clinoid, it penetrates the dura, giving off in this region its first large branch, the ophthalmic. Through the ophthalmic the internal carotid has extensive anastomotic connections with the external carotid of the same and opposite sides (Walsh and co-workers).[5] Continuing between the optic and oculomotor nerves, the carotid divides into the anterior and middle cerebral vessels above and lateral to the optic chiasm. Just before this bifurcation, it receives the posterior communicating artery which runs above and roughly parallel with the oculomotor nerve to connect with the posterior cerebral artery. The circle of Willis is completed anteriorly by the anterior communicating artery which connects the two anterior cerebral vessels.

While the ophthalmic is the first major branch of the carotid after its bifurcation in the neck, it should be remembered that six vessels or sets of vessels are described as arising from the petrous or cavernous carotid before it reaches the ophthalmic. These are the caroticotympanic, the vidian, the cavernous, the hypophyseal, the semilunar, and the anterior meningeal branches. In order to understand the pathogenesis of aneurysms in this portion of the carotid artery, it is important to remember these smaller branches.

The oculomotor nerve, as it passes forward from its origin in the brain stem toward the dura of the cavernous sinus, parallels the posterior communicating artery on its medial side (Whitnall).[6] Although it reaches the dura at the posterior clinoid, it has been shown by Laphart (1925) (see Whitnall, pp. 323 and 322)[6] that the nerve is excluded from the sinus proper until after it has dipped beneath the anterior clinoid process. In the cavernous sinus wall the nerve lies above and somewhat lateral to the carotid. It passes forward below the ophthalmic nerve and below and medial to the trochlear to enter the orbit through the superior orbital fissure (Whitnall). Within the cavernous sinus the oculomotor receives sympathetic fibers from the carotid plexus as well as sensory fibers from the first division of the trigeminal (Whitnall, p. 334).[6]

The trochlear nerve lies just lateral and inferior to the oculomotor in the cavernous sinus wall. It is somewhat more laterally placed with reference to the carotid but could be involved, alone or in conjunction with other nerves, by a suitably located aneurysm. In the anterior part of the sinus it crosses and is connected with the third nerve as it rises to a point above it and parallel with the ophthalmic at this level. It, too, receives communications from the cavernous plexus and from the ophthalmic division of the trigeminal. The sixth cranial nerve, as it passes through the cavernous wall, lies inferior to the artery and to the above described nerve complex.

It is seen, therefore, that aneurysms of the intracranial and cavernous portions of the carotid artery, as well as of the posterior communicating, the proximal portion of the middle cerebral, and the proximal portion of the anterior cerebral, may well be in a position to affect the nerves of ocular motion, particularly the oculomotor. The vessels named are frequent sites of saccular aneurysms.

Congenital saccular aneurysms arise from weak areas in the walls of intracranial arteries where there has been incomplete involution of the embryonal vessels (Dandy),[1] or from defects at the point of bifurcation of larger intracranial vessels (Forbus).[3] Although miliary aneurysms due to atheromatous changes in the walls of smaller blood vessels in older people are said to occur somewhat more frequently (Walsh and co-workers),[5] it is the congenital aneurysm which is of greatest clinical interest from both the diagnostic and therapeutic standpoints.

Saccular aneurysm of the intracranial vessels is a fairly common malady. Dott,[2] in a review of the literature in 1933, estimated that the lesion was found in approximately one in 700 consecutive postmortem examinations. Many of these, however, had neither produced symptoms nor contributed to the cause of death. Intracranial aneurysms arise most frequently from the carotid artery or its principal connections. McDonald and Korb[4] described 774 of 1,023 aneurysms as originating on these vessels; 480 were on the carotid or the anterior circle of Willis. Fortunately, it is in these locations that they are most easily diagnosed by clinical signs, including the ones under discussion, as well as by angiography. They are the ones which offer the best opportunity for successful treatment.

Arteriovenous fistula usually is a more dramatic disease since in the acute stage there is added to the signs of cranial nerve involvement a pulsating protrusion of the eye which becomes, therefore, the prominent sign of this potentially serious lesion. Chronic fistulae, however, may simulate many of the signs and symptoms of saccular aneurysm and therefore should be mentioned.

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